AMENDMENTS TO THE CLAIMS

1. (Currently amended) A method of identifying genetic mutations that are associated with ~~ataxic neurological disease~~ adult onset cerebellar ataxia in a human subject, said method comprising:

(a) determining a first nucleic acid sequence of a human protein kinase C gamma gene from a first human subject exhibiting adult onset cerebellar ataxia;

(b) identifying a difference between the first nucleic acid sequence from the first human subject exhibiting adult onset cerebellar ataxia and SEQ ID NO:3, wherein the difference alters the amino acid sequence encoded by the human protein kinase C gamma gene; and

(c) confirming that the difference identified between the first nucleic acid sequence and SEQ ID NO:3 is a genetic mutation associated with adult onset cerebellar ataxia by co-segregation analysis comprising determining that the identified nucleic acid sequence difference is also present in a plurality of human subjects exhibiting adult onset cerebellar ataxia and is absent in a plurality of human subjects not exhibiting adult onset cerebellar ataxia.

2. (Currently amended) The method of Claim 1 wherein the first nucleic acid sequence from said first human subject is determined by amplification of ~~portions~~ at least a portion of the human protein kinase C gamma gene from genomic DNA isolated from said human subject to produce an amplified DNA and sequencing said amplified DNA.

3. (Canceled)

4. (Currently amended) The method of Claim 1 wherein said ~~cosegregation~~ co-segregation analysis comprises a method selected from the group consisting of direct

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sequencing, sequencing PCR-amplified DNA, single stranded conformation analysis, allele-specific PCR and restriction fragment length polymorphism.

5. (Currently amended) The method of Claim 4 wherein said ~~cosegregation~~ co-segregation analysis comprises sequencing PCR-amplified DNA.

6. (Currently amended) The method of Claim 4 wherein said ~~cosegregation~~ co-segregation analysis comprises restriction fragment length polymorphism analysis.

7-42. (Canceled)

43. (Withdrawn – currently amended) The method of Claim [[2]] 1, wherein the ~~portions of nucleic acid sequence that are amplified comprises at least one of~~ first nucleic acid sequence is a coding region of the human protein kinase C gamma gene selected from the group consisting of exon 1 ~~(nucleotides 440 to 609 of SEQ ID NO:3)~~; exon 2 ~~(nucleotides 1108 to 1139 of SEQ ID NO:3)~~; exon 3 ~~(nucleotides 2106 to 2188 of SEQ ID NO:3)~~; exon 4 ~~(nucleotides 7583 to 7694 of SEQ ID NO:3)~~; exon 5 ~~(nucleotides 7831 to 7962 of SEQ ID NO:3)~~; exon 6 ~~(nucleotides 9619 to 9775 of SEQ ID NO:3)~~; exon 7 ~~(nucleotides 10454 to 10588 of SEQ ID NO:3)~~; exon 8 ~~(nucleotides 10933 to 11020 of SEQ ID NO:3)~~; exon 9 ~~(nucleotides 11307 to 11336 of SEQ ID NO:3)~~; exon 10 ~~(nucleotides 15904 to 16056 of SEQ ID NO:3)~~; exon 11 ~~(nucleotides 16385 to 16573 of SEQ ID NO:3)~~; exon 12 ~~(nucleotides 18178 to 18269 of SEQ ID NO:3)~~; exon 13 ~~(nucleotides 18364 to 18426 of SEQ ID NO:3)~~; exon 14 ~~(nucleotides 18556 to 18694 of SEQ ID NO:3)~~; exon 15 ~~(nucleotides 21018 to 21098 of SEQ ID NO:3)~~; exon 16 ~~(nucleotides 22580 to 22687 of SEQ ID NO:3)~~; exon 17 ~~(nucleotides 24262 to 24402 of SEQ ID NO:3)~~; [[or]] and exon 18 ~~(nucleotides 24652 to 24840 of SEQ ID NO:3)~~.

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44. (Currently amended) The method of Claim [[43]] 1, wherein the ~~portion of SEQ ID NO:3 that is amplified~~ first nucleic acid sequence comprises exon 4 ~~(nucleotides 7583 to 7694 of SEQ ID NO:3)~~ of the human protein kinase C gamma gene.

45. (Currently amended) The method of Claim 1, wherein the mutation associated with adult onset cerebellar ataxia ~~neurological disease~~ is selected from the group consisting of a missense mutation, a deletion mutation, and an insertion mutation~~, a splicing site mutation, and a mutation that results in loss of expression of the protein kinase C gamma gene encoded by SEQ ID NO:3~~.

46. (Previously presented) The method of Claim 45, wherein the mutation is a missense mutation.

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